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Radiofrequency Exposure and Human Cancers: Elwood's Response

I thank Hocking for his interest in my review (*1*). In regard to his own study (*2*), I put more emphasis on the incidence than the mortality results for several reasons. The interpretation of the mortality results is more complex, requiring control for confounding by prognostic factors (such as stage at diagnosis and precise age) as well as by risk factors for incidence. The difference between the relative risks for incidence and for mortality is not statistically significant, and of course the two results are not independent. The incidence results are also more useful because they can be compared with those of another study. The discussion in the paper by Hocking et al. (*2*) is almost all on the incidence relationship. The suggestion that radiofrequency radiation (RFR) exposure is related to adverse survival is a new hypothesis generated from these results and, as far as I know, has not been assessed in other studies.

The comparison of the two studies of childhood leukemia in Sydney, Australia (*2–4*), involves a comparison of concepts. In his letter, Hocking claims that the original hypothesis for these studies was that the leukemia rate in the three areas close to the TV towers would be different from the rate in the six areas farther away; as stated in my review (*1*), his statistical analysis depends on this comparison. However, in my opinion, the original hypothesis is epidemiological—whether there is an increased cancer incidence (and mortality) in children exposed to RFR from TV towers; this is given as the objective in the first paper by Hocking et al. (*2*). The use of a statistical design that compares two sets of areas is one way to assess this. This approach is not unreasonable but ignores the information provided by the comparison of each individual area. Such data are relevant to the assessment of the consistency of any association, which is an important aspect in assessing causality. I was surprised that the results by individual municipality, which Hocking et al. had available, were not given in the original paper (*4*), as I believe they affect the interpretation. The subsequent analysis showed that the excess was seen in only one of the three areas close to the TV towers (*3*). Because of statistical variability, this does not rule out the general association seen by Hocking et al., but it shows inconsistency and weakens the argument that the association seen is caused by RFR from the TV towers rather than from any other cause.

In the Polish military study (*5*), the published report states that information on possible carcinogenic factors and RFR exposure was available for cancer cases from hospital records, in addition to data from other sources available for all personnel. This raises the possibility of systematic bias, as some information on exposure is available only for affected subjects. This potential bias has been noted independently in another detailed epidemiologic review (*6*). In regard to the U.S. Navy study (*7*), Hocking emphasizes the major weakness of the study, which I have noted. I agree that this study is very limited in exposure information.

In the case–control study of brain cancers, Thomas et al. (*8*) found a significant excess risk in electronics workers with no exposure to RFR, and no excess risk in those exposed to RFR who were not electronics workers. There was an increased risk in electronics workers who were also exposed to RFR, but this risk was lower than the risks for all electronics workers. Although this may be consistent with some complex promotional effect, the more parsimonious explanation is that the increased risk in electronics workers is due to some exposure other than RFR.

In his letter, Hocking refers to a New Zealand environment court case (*9*) that concerned a proposed Telecom cell phone transmitter site near a school. I appeared as an expert witness for Telecom, and he appeared as a witness for the school. My published review (*1*) was developed at the same time as my written evidence, but was not submitted until after the case in order to benefit from legal review as well as from scientific peer review. The legal hearing has resulted in a detailed judgment in favor of Telecom (*9*). In his judgment, Judge Jackson commented on each of the several expert witness submissions. He noted that "Elwood's evidence was carefully constructed and balanced" (*9*).

In summary, although the points raised by Hocking are worthy of note, I do not agree that any of them represent "important omissions" in my review paper.

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Comments on "What Is a Tumor Promoter?"

In the August issue of *Environmental Health Perspectives*, Raymond Tennant (*1*). shared his

> perspective on how the identification of tumor promotion relates to the assessment of human health risk from environmental carcinogens.

I would like to reply to several of his statements. Although a complete reanalysis of his perspective is beyond this letter, I recommend additional reading (*2–6*). My comments are based on looking at the multistep, multimechanism process of carcinogenesis from a completely different paradigm, based on different assumptions.

Tennant (*1*) states that

> The role of the tumor-promoting agents has not been so specifically defined, even in the most well-studied mouse skin model.

It has been known for over 20 years that a testable hypothesis exists, based on a specific cellular mechanism; this hypothesis is supported by data derived from molecular oncological, biochemical, cellular, and now knockout mouse data (*2,7*). This mechanistic model, namely, the reversible inhibition of gap junctional intercellular communication (GJIC), is as complete, if not more so, than our detailed mechanistic understanding of "initiation," which is assumed to be related to DNA damage and mutagenesis.